Erwin Chemerinsky (*pro hac vice*)
echemerinsky@law.berkeley.edu
Claudia Polsky (CA Bar No. 185505)
cpolsky@law.berkeley.edu
U.C. BERKELEY SCHOOL OF LAW
Law Building
Berkeley, CA 94720-7200
Telephone: 510.642.6483

Elizabeth J. Cabraser (CA Bar No. 83151)
ecabraser@lchb.com
Richard M. Heimann (CA Bar No. 63607)
rheimann@lchb.com
LIEFF CABRASER HEIMANN &
BERNSTEIN, LLP
275 Battery Street, 29th Floor
San Francisco, CA 94111
Telephone: 415.956.1000

Anthony P. Schoenberg (CA Bar No. 203714)
tschoenberg@fbm.com
Linda S. Gilleran (CA Bar No. 307107)
lgilleran@fbm.com
FARELLA BRAUN + MARTEL LLP
One Bush Street, Suite 900
San Francisco, CA 94104
Telephone: 415. 954.4400

Attorneys for Plaintiffs and the Certified Classes

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

NEETA THAKUR, et al.,

Plaintiffs,

v.

DONALD J. TRUMP, et al.,

Defendants.

Case No. 3:25-cv-4737

DECLARATION OF RHONDA VOSKUHL

DECLARATION OF RHONDA VOSKUHL

I, Rhonda Voskuhl, declare as follows:

1. I have personal knowledge of the facts contained in this declaration and, if called as a witness, could and would testify competently to those facts.

2. I am a Professor of Neurology at the University of California Los Angeles School of Medicine. I hold the Jack H. Skirball Chair and have served as the Director of the UCLA Multiple Sclerosis Program since 2000. I am also a Faculty Neurologist for the UCLA Comprehensive Menopause Program. In the neurology clinic at UCLA, I treat patients with multiple sclerosis ("MS") and menopausal women with cognitive issues.

3. I earned an M.D. from Vanderbilt Medical School and subsequently completed neurology residency at the University of Texas Southwestern and a fellowship in neuroimmunology at the National Institutes of Health ("NIH"). In 1995, I joined the faculty of UCLA as an Assistant Professor in the Department of Neurology. I was promoted to Associate Professor in 2000 and full Professor in 2004. From 1995 to 2000, I served as the Scientific Director of the UCLA Multiple Sclerosis Program.

4. I have won numerous national and international awards for my work in neuroprotective treatment drug discovery, most recently including the John Dystel Prize in Multiple Sclerosis, 2024, from the American Academy of Neurology and the National MS Society, the most prestigious award in the field of MS. I was also awarded the Rachel Horne Prize for Women's Research in Multiple Sclerosis, 2023, from the European and American Committees for Treatment and Research in MS.

5. My research at UCLA focuses on determining how sex hormones and sex chromosomes cause sex differences in the onset and severity of neurodegenerative diseases. I am an internationally recognized expert in sex differences research, demonstrating protective effects of estrogen and testosterone treatment in preclinical models, which I translated to five clinical trials in patients. My lab was the first to show that estrogen receptor (ER) alpha and ER beta ligands act through distinct mechanisms to induce neuroprotection. I also discovered that an X chromosome gene (the histone demyethylase *Kdm6a*) increases neuroinflammation. In addition,

my lab was the first to use brain cell-specific and region-specific transcriptomics to investigate the molecular basis for disability-specific disease progression in MS. My lab also investigates the role of brain aging on neurodegeneration, identifying a sex hormone by age interaction whereby being estrogen deficient and midlife combine to induce cognitive decline, dorsal hippocampal atrophy, glial activation, and synaptic loss. The goal of my research is to use a brain region-specific, cell-specific, and sex-specific approach to identify neuroprotective treatment targets, then design clinical trials to repair neurodegeneration which are optimally tailored for sex and age.

6. I have authored more than 200 publications, including in prestigious journals such as *Nature*, *Lancet Neurology*, and the *Proceedings of the National Academy of Sciences.* These include showing how estrogen protects the brain from cognitive decline and regional brain atrophy during health and disease. My career was profiled in the February 2024 issue of *Lancet Neurology*, the preeminent neurology journal in the world.

7. I have been the recipient of a total of 79 research grants for my work from governmental and private sources. I have received 40 research grants from the NIH, many of which have been multi-year awards providing funds for 2, 3 4 or 5 years. Grants have provided continuous funding from 1997 to present. Throughout the last 28 years, I have never before received a notice from NIH freezing or rescinding previously awarded funding, up until NIH suspended a previously awarded grant funding active research work, as detailed below.

8. A true and correct copy of my biographical sketch is attached as Exhibit A.

Application for Grant Funding from the NIH

9. On July 8, 2022, I submitted, in conjunction with the UCLA Office of Contract and Grant Administration, an Application for Federal Assistance to the NIH for a project titled "Neurodegeneration Underlying Distinct Disabilities in Multiple Sclerosis Using a Cell-Specific, Region-Specific, and Sex-Specific Approach" (the "R35 Application").

10. The Project Narrative for the R35 Application explained:

> This R35 proposal will: 1) Extend our cell-specific and region-specific transcriptomics in astrocytes and oligodendrocytes to microglia and neurons, with cell:cell interactions revealed in mice double-labelled to show gene expression changes in two distinct cell

types in the same region in the same mouse, and 2) Determine if there are effects of sex and/or age on the most differentially expressed cell-specific and region-specific pathways. In summary, this R35 proposal takes our research to the next level: Identifying sex by age interactions in cell-specific and region-specific transcriptomics, neuropathology, and substructure atrophy on MRI.

The greatest unmet need in multiple sclerosis (MS) is to develop novel treatments targeting cells and processes within the central nervous system (CNS) to confer neuroprotection and repair disabilities, in not only relapsing remitting MS, but also in secondary progressive MS. A "one size fits all" neuroprotective treatment approach in MS will not work, since 1) MS patients are heterogenous regarding which disabilities are predominant, 2) being female versus male impacts rates of disability progression, and 3) aging corresponds with disability progression. This R35 will use a cell-specific, region-specific, and sex-specific approach to discover neurodegenerative targets optimized for each disability in MS models in females and males during young adulthood and aging.

11. The proposed project (the "R35 Project") built on work that I and my team had done over years:

a. My lab discovered sex chromosome effects in the immune system in MS models: we identified a gene on the X chromosome (*Kdm6a*) that escapes X-inactivation in CD4+T lymphocytes as a mechanism for increased susceptibility of females to autoimmune disease. Focusing on the CNS, we showed that in contrast to XX conferring an increase in autoimmunity, XY confers an increase in the neurodegenerative response to the same autoimmune attack. Indeed, my lab was the first to show an effect of sex chromosome complement in the CNS in any neurodegenerative disease model.

b. My lab used a cell-specific and region-specific transcriptomics approach to identify novel mechanisms underlying regional neuropathology in MS models and MS autopsy tissues. While this approach had been used in astrocytes during health, my lab was the first to use a cell-specific and region-specific transcriptomics approach in any neurodegenerative disease model.

c. Estrogens were known to be neuroprotective through actions on estrogen receptors (ERs) for decades, however which cell in the CNS mediated this neuroprotection in vivo remained unknown. My lab created cell-specific knock outs of ER alpha and ER beta to determine which CNS cell mediated neuroprotection in vivo. My lab was the first to identify which cell is responsible for estrogen mediated neuroprotection in vivo in any neurological

disease model.

12. The R35 Application requested $7,307,976.00 for an eight-year period (4/1/2023 – 03/31/2031); I was identified as the Project Director and Principal Investigator on the Application. The proposal would fund salaries for myself, four co-investigators, one graduate student, one senior lab technician, and one MRI lab technician.

13. A true and correct copy of the R35 Application dated July 8, 2022, is attached as Exhibit B.

Award of Grant Funding for R35 Grant

14. On May 8, 2023, the Department of Health and Human Services ("DHHS"), NIH, National Institute of Neurological Disorders and Stroke issued a Notice of Award, Federal Award Identification Number R35NS132150 (the "R35 Grant Award"), approving the R35 Application. The R35 Grant Award was awarded for a total of eight years, as sought in the R35 Application, for an amount of $876,448 for the May 15, 2023-April 30, 2024, budget period, and additional awards of $913,497 for the next seven years. The statutory authority for the award was "42 USC 241, 42 CFR 52."

15. R35 Awards are only granted to approximately ten neuroscience researchers per year across the United States.

16. My team and I began work on the R35 Project in May 2023, executing the research studies outlined in the grant application. NIH approved continuing funding for the R35 Project in each of the subsequent years.

17. During the initial two years of the eight-year R35 Grant Award, insights have been discovered in my lab which are clinically significant for patients.

a. My lab discovered that the resident immune cell of the CNS, the microglia, overexpresses the X chromosome gene (*Kdm6a*) in females, and this causes more brain inflammation. Pharmacologic blocking of this using metformin, a widely used diabetic drug with anti-aging and neuroprotective properties, worked better in females than in males. This has implications for the efficacy of metformin treatment in women and men today.

b. The supportive cell of the CNS, the astrocyte, was discovered to confer

neuroprotection during estrogen treatment in otherwise healthy females during midlife aging. Treatment specifically targeting estrogen receptor beta in astrocytes prevented cognitive decline, regional atrophy on brain MRI, and neuropathology. Gene expression changes in energy metabolism within astrocytes in the menopause model aligned with gene expression changes in brains of humans (menopausal women). Estrogen receptor specific targeting identifies a window of opportunity to stimulate estrogen receptor beta for protection in brain, while minimizing stimulation of estrogen receptor alpha in breast to reduce breast cancer risk.

c. My lab's findings suggest a critical balance between sex chromosomes and sex hormones in health and disease. A female sex chromosome (XX) gene drives neuroinflammation and neurodegeneration during aging, MS, and Alzheimer's Disease (AD). This is why women are more likely to get MS and AD. Balancing this, a female sex hormone (estrogen) is anti-inflammatory and neuroprotective in women at ages before menopause. However, when estrogen mediated neuroprotection is lost abruptly and permanently at menopause in otherwise healthy women (mean age 51 years), 60-70% experience cognitive domain specific symptoms. Brain regional changes on MRI align with cognitive complaints. Further, women with MS and AD have disability worsening and/or disease onset, respectively, at menopause.

d. The research in my lab resulted in me being the lead inventor on several UCLA patents in the U.S. and Europe that identify a novel estrogen treatment approach to prevent cognitive decline in aging, MS, and AD. The U.S. patents were licensed from UCLA by CleopatraRX, and blisterpacks of this patented hormone treatment were designed for menopausal women (PearlPAK). This new treatment is now commercially available across the U.S.

e. The suspended R35 research plan also includes finding neuroprotective treatments for men. Work in my lab is determining the balance between the role of male sex chromosome genes (XY) and male sex hormones (testosterone) in neuroinflammation and neuroprotection. The goal is to prevent neurodegeneration during andropause, when testosterone levels decrease gradually in men from age 30 to 70 years.

18. Overall, my work in animal models has tangible relevance to human health and

disease. I have designed and carried out several clinical trials, with two more now in planning stages. I am known for, indeed I was profiled in, the February 2024 issue of *Lancet Neurology*, entitled "Bedside to Bench to Bedside" research. This means clinical observations in patients ("Bedside") lead to treatment target discovery in animal models ("Bench") which lead to testing new treatments in human clinical trials ("Bedside").

19. A true and correct copy of the May 2023 R35 Grant Award is attached as Exhibit C. True and correct copies of additional Notices of Award, authorizing continuing funding, pursuant to the R35 Application and R35 Grant Award, issued in April 2024 and June 2025, are attached as Exhibits D and E.

Suspension of Grant Funding

20. On July 31, 2025, the UCLA Chancellor, Dr. Julio Frenk, received a letter from Jon Lorsch, the Acting Deputy Director for Extramural Research at NIH (the "Notice of Award Suspensions"). The Notice of Award Suspensions indicated that NIH was "hereby suspending the attached list of grant awards" and that UCLA researchers "must cease all activities on the awards and immediately discontinue drawing down funds from the Payment Management System (PMS) for any expenses incurred after receipt of this letter." The letter further stated that "under 45 CFR § 75.372 and 45 CFR § 75. 373, NIH may move to terminate an award" for various reasons. A copy of Notice of Award Suspensions was later forwarded to my email, together with a spreadsheet of suspended NIH grants. My R35 Grant Award is listed on the spreadsheet. A true and correct copy of the Notice of Award Suspensions is attached as Exhibit F.

21. On August 1, 2025, Tracey Fraser from the UCLA Office of Contract & Grant Administration sent an email instructing me to "***immediately stop incurring costs/expenditures on the grant(s) referenced above*** effective July 31, 2025." This "Stop Work Notice" was initially mistakenly sent to an inactive email address, rvoskuhl@ucla.edu, and a copy was not forwarded to my correct email address, rvoskuhl@mednet.ucla.edu, until later. A true and correct copy of the Stop Work Notice is attached as Exhibit G.

22. I first learned of the suspension of my grant on August 4, 2025, when I received an email from S. Thomas Carmichael, the Chair of the Department of Neurology at UCLA, inviting

me and the 23 other faculty in our department affected by grant suspensions to discuss our "lost grants," the "loss of supplies and other support for research" and the "substantial negative effect for faculty and their research programs." A true and correct copy of Dr. Carmichael's email is attached as Exhibit H.

23. I was not offered any reason for the suspension of my grant; any means of appealing this suspension; or informed of any other action I could take to reinstate the grant.

Harm Suffered from Termination of Grant Funding

24. I and my project team have suffered immediate harms as a result of NIH's actions in suspending this grant. These harms are continuing. Specifically:

a. The R35 Grant Award funded research conducted by myself, as well as by four co-investigators: A Faculty Specialist in genetic analysis and bioinformatics, a Faculty Professor in statistical genomics and bioinformatics, a Faculty Professor in neuroimaging, and a Faculty Research statistician. The Award also funded my lab's senior technician, an MRI lab technician, and graduate student researchers. It also funded part time work for 3 to 4 UCLA undergraduates each year who spend 10-15 hours per week working in my lab, as well as 1 to 2 undergraduates who work full time during their summer breaks.

b. My lab's only NIH grant is the R35 that was suspended. Importantly, an R35 from NIH NINDS precludes a researcher from applying for funding to NINDS for any other basic research grants during the duration of the R35 eight-year funding period. The R35 is intended to allow Professors with a track record of extraordinary success to have substantial and stable funding for eight years in order to address large scope questions. This is more efficient than spending substantial time and resources applying for numerous smaller, shorter-term grants in a piecemeal fashion. As a result of loss of my one and only NIH funding source, my lab is no longer able to purchase supplies for our experiments. I have informative, genetically engineered mice that took over 3 years to generate, since they model a disease aspect and/or deletion of a critical gene in neuropathogenesis. These mice will soon be lost, as will my ability to determine reasons for neurodegeneration in females and males, at adulthood and midlife aging. Based on my track record, my lab's research using them would likely have identified a treatment target to

provide rationale for design of a clinical trial tailored for either women or men at young adulthood or midlife with MS, cognitive decline in otherwise healthy people, or Mild Cognitive Impairment, a prelude to Alzheimer's Disease. I will soon have to let my staff go due to lack of funding. This team of researchers has taken two decades for me to gradually build. They have complex synergistic skills in neuroimmunology, neurogenetics, neuroendocrinology, neuropathology, and neurobehavior. An intangible is that we are an efficient and effective team. Rebuilding such a team would take me at least ten years. Research in my lab will grind to a halt. Any temporary pause, even for a few months, has lasting consequences in terms of our research productivity, our laboratory's output, and the publications we produce.

c. My co-investigators, who have highly specialized training but are more junior in their careers, will be harmed by a gap in publications, which will negatively impact their career progression and ability to secure future funding for their research. Without funding, I will not be able to retain or recruit graduate student researchers, which will harm graduate student training and career prospects. On average, I take in two undergraduates per year when they are UCLA sophomores or juniors who stay until they graduate, resulting in 4-6 at any given time. They learn how to do research, give presentations, win student awards, and become a co-author on publications. My research lab is a launch point for their career as they plan and apply for either M.D., Ph.D., or combined M.D., Ph.D. programs across the country. Each year, one will stay in my lab and work for a gap year, as they apply and get accepted to graduate school. Shutting down research in my lab will shut down the hopes and dreams of countless undergraduates. My lab is unique since it is truly translational, basic science applied to discovery of new treatments. Students love this. They will be lost with this suspension.

d. A pause in our research negatively impacts my subfield of neurology. My team will be unable to share our research findings at conferences and in scientific publications. I also do many media interviews about our science to inform and educate the public on our latest findings in the context of current knowledge in the field. This will end, since no more funding means no more findings. Since I am training the next generation of neuroscience researchers, including young faculty, postdoctoral fellows, graduate students and undergraduates, the future of

this field of research will be harmed by an indefinite pause in training. This goes beyond neurology to include the gynecologists and internists that I am training about the neurology of menopausal women with cognitive issues.

e. The U.S. public, which ultimately funds NIH grants, will also lose much of the value of their investment if my NIH grant is indefinitely suspended. Our research has already generated new insights into the molecular basis of disability-specific disease progression in MS. Multiple sclerosis affects nearly one million people in the United States, and since it usually starts in their 30s, patients must manage an approximate 50-year burden of disease. Work supported by this grant is aimed at developing novel treatments targeting cells and processes within the central nervous system to confer neuroprotection and repair disabilities for MS patients. Also, our research has already generated new insights into the molecular basis for cognitive decline during aging in people otherwise healthy, namely menopausal women and andropausal men. These issues impact everyone who lives long enough to go through menopause and andropause (over age 50). We already have UCLA licensed patents, with a novel hormone replacement therapy (HRT) now commercialized and on the market for menopausal women across the country as well as in the UCLA Comprehensive Menopause Program. Its foundation is in my lab's past NIH funded basic research and repurposing our findings in MS showing improved cognition and reduced regional brain atrophy in women with MS. NIH's withheld funding threatens the loss of clinically relevant research discoveries as well as current and future treatments.

I declare under penalty of perjury under the laws of the State of California and the United States that the foregoing is true and correct.

Executed this 22nd day of August, 2025, in Los Angeles, California.

DocuSigned by:
8833E07DB6D646A...
Rhonda Voskuhl